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Effect of Lesion of the Lateral Geniculate Body on the EEGs and Photically Evoked Potentials in the Cerebral Visual, Somatosensory and Association Areas of Unanesthetized Cat

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Visually evoked potentials and background EEGs in the cerebral visual, somatosensory, and association areas (lateral suprasylvian gyrus), as well as in the lateral geniculate body (GL) and mesencephalic reticular formation were studied before and after unilateral destruction of the GL of unanesthetized cat. Autocorrelograms and power spectra of background EEGs in various areas mainly showed activities of less than 5 c/s. Unilateral GL destruction caused considerable depression of the ipsilateral EEG activities and mild depression in the contralateral EEGs in most regions except the somatosensory area, where some augmentation was observed. Unilateral putamen destruction resulted in augmented EEG activities in various areas. GL activity seemed to augment EEG activities in the cerebral visual and association areas, while it seemed to depress the activity in the somatosensory area.

Summation average of evoked potentials in the association area likewise manifested quite similar diphasic deflections within 130 msec after flashing light stimulus. The initial positive deflection P_1 led bipolarily in the medial visual association area was found to be longer in duration than in the medial visual area. Peak latencies of the second positive-negative deflection (P_2N_2) and third positive deflection P_3 in the association area were about 10 msec longer than those in the visual area and this was proven to be statistically significant. Amplitudes of the initial and second diphasic deflections in the medial visual area were higher than those in the posterior visual and medial association areas, whereas amplitudes of the third and fourth deflections in the medial visual area were lower than those in the association area.

Almost complete destruction of the GL resulted in disappearence of the initial and succeeding diphasic deflections, and there remained a slow negative wave with a crest at about 70 msec after the flashing stimulus which presumably had been masked by the above deflections prior to GL

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destruction. After partial destruction of the GL, the initial and succeeding deflections were sustained, but prolonged in their peak latencies and decreased in their amplitudes. It was suggested, therefore, that the association area (medial lateral suprasylvian gyrus) may have intimate interaction with the lateral geniculate body, and that the slow negative wave following the initial positive deflection may have been brought out through the indirect nonspecific projection system.

INTRODUCTION

Many reports have been published on the visually evoked potential (VEP) of cat elicited by flashing light stimulus.[5,7,8,12,21] Such authors as BRAZIER (1957)[3], HIROTA (1965)[9], STEINBERG(1965)[18], TAGAWA (1966)[19], NORTON and JEWETT (1967)[14], etc. have applied computer techniques to the analysis of various response components of the VEP in the visual area brought out during the period 200 msec to 750 msec after flashing light stimulus. It was demonstrated by BIGNALL et al. (1966)[5] that the VEP is brought out not only in the primary visual area, but also in the secondary visual area (Area 18, OTUKA and HASSELER 1962)[16] and in the lateral suprasylvian gyrus (association area), to which direct input from the lateral geniculate body (GL) has been reported by VASTOLA (1961)[22], etc. Further, the VEP produced in the lateral suprasylvian gyrus has been found to remain intact even after destruction of the nonspecific portion in the thalamus (BIGNALL 1966[5]).

In this paper, therefore, an analysis of the VEP in the above mentioned association area was done in relation to that in the lateral gyrus (visual area) of unanesthetized, immobilized cat by the application of the summation average method using the computer, before and after destructian of the lateral geniculate body (GL). In addition, the influences of GL destruction on EEG background activities were also observed in terms of autocorrelogram and power spectra.

METHODS

22 adult cats weighting 2.3–4 Kg were used. After tracheal canulation under ether anesthesia, the animal was mounted on JOHNSON's type stereotaxic instrument. Holes of about 1 mm diameter were made on the scalp. Following this surgical procedure, gallamine triiodoethylate (Flaxedil) was administrated intraperitoneally, 20 mg/Kg body weight, to immobilize the cat and artificial respiration with oxygen mixed air was performed at a rate of 24 per minute. Silver-ball tipped electrodes, 0.5mm in diameter, were placed as surface electrodes on the dura in the bottom of the holes. Four, three and one surface electrodes were placed in the lateral gyrus (L_1, L_2, L_3, and L_4) lateral suprasilvian gyrus (SS_2, SS_3 and SS_4) and posterior sigmoid gyrus

(PS), respectively. The approximate stereotaxic coordinates of these surface electrodes were PS (A: 22.0, L: 6.0), L_4 (A: 16.0, L: 2.0), L_3 (A: 6.0, L: 2.0), L_2 (A: −2.0, L: 2.0), L_1 (A: −6.0, L: 4.0), SS_3 (A: 6.0, L: 9.0), SS_4 (A: 16.0, L: 9.0) and SS_2 (A: −2.0, L: 9.0), respectively. Bipolar depth electrodes of stainless steel, insulated except at their tips, were inserted 2−3 mm apart aimed at the lateral geniculate body (GL) (A: 6.0, L: 10.0, H: +3.0). GL was destroyed by delivery of a current of 5−6 mA through the depth electrodes for 5−6 minutes in most instances, and a current of over 10 mA for more than 10 minutes in some instances. In control experiments, depth electrodes were inserted into the putamen (A: 12.0, L: 10.5, H: −1.0) of two cats to destroy the putamen using the same current as that for GL destruction. Electroencephalographic potentials in various areas on the cortical surface and in the lateral geniculate body were led monopolarily from the surface silver-ball tipped electrodes and one of the depth electrodes, respectively. The reference silver electrode was inserted into the neck muscle. The location of the tips of the depth electrodes and the region destroyed were checked histologically by KLÜVER-BARRERA's staining method[11] after sacrificing the animal at the end of the experiment. Flashing light was delivered through the photic stimulator (PS 101, San'ei-Sokki Co.) at 1.6 or 2.0 sec intervals to both eyes, which had been dilated by 0.5% atropin sulphate. The flashing light from the strobo flash bulb (FT-100, Mazda) was of about 6000°K bluish, white daylight, 1 Whatt/sec and about 100 μsec duration. The bulb was placed 50 cm from the eyes. A frosted glass plate and an iris of 100 mm diameter were placed in front of the bulb to diffuse the light and to fix its intensity. For greater diffusion, a white cotton curtain was hung between the iris and the eyes. If the intensity of the diffused flashing light passing through the iris of 5 mm diameter is taken as 0 on a logarithmic scale, then the relative intensity of the flashing light through the iris of 100 mm diameter was 2.6 as measured by photovalve and CRO (see SATO and KITAJIMA 1965[17]). Masspotentials in various sites and the stimulus signal were recorded onto 1/4 inch magnetic tape by the PWM and FM methods, respectively, through 8-channel polygraph (RM-150 Nihon Kohden Co.) and 8-channel Data Recorder (SPRA-48, Shiroyama Tsûshin or SDR-41 Nihon Kohden). EEG records were monitored by inkrecords of the polygraph and two four-beam CROs (VC6-Nihon Kohden Co.), while the stimulus signal was monitored by one beam synchroscope (SS-5022, Iwasaki Tsûshin, Co.). The averaged visual evoked potentials (VEP) by summation technique were obtained by feeding the play back currents from the magnetic record into the Digital Computer for Data Processing (ATAC-401 or ATAC-501−20, Nihon Kohden Co.).

Autocorrelograms and power spectra of EEGs were computed by

HITAC 5020 (Computer Center, Tokyo University) or FACOM 270−20 (Shionogi Computer Center, Osaka) on 600 discrete data obtained at every 1/60 sec (=16.7msec) from an EEG record of 10 sec length. Autocorrelograms of EEGs were also obtained by ATAC−501−20.

RESULTS

It was confirmed by histological examination that the GL had been almost completely damaged in two cats. In twelve cats it was partially destroyed, of which five had partial destruction of the optic radiation (OR) and/or optic tract (OT), while the other seven cats had destruction of suprasylvian gyrus around the depth electrode. In six cats, the GL was intact with damage of the optic radiation only or of the OR and suprasylvian gyrus. In two cats, putamen destruction was performed as the control experiments.

1. *Influences of GL lesion on background EEG activity.*

Fig. 1A shows an example of a histological section of brain (cat#71) showing destruction of the dorsolateral site (A layer) of the GL and white matter of the cerebrum. EEG ink records, before and after the destruction, in the posterior sigmoid (PS), lateral (L) and lateral suprasylvian gyri (SS), lateral geniculate body (mono-(GL(M)) and bipolar leads (RF(B)) are depicted in Fig. 1B. Five minutes after destruction of the right GL, rhythmic activities which had been seen in all of the above EEGs were depressed, and bipolar records of GL showed irregular, slow fluctuations. The changes in EEGs which were still unstable five minutes after destruction gradually became stable with elapse of time, and after twenty minutes they seemed to have become sufficiently stable.

Twenty minutes after destruction, EEG activities, e.g. 11−12 c/s activities, in the PS had recovered adequately, whereas those in other regions had not. Recovery of EEG activities in L and RF(B) was relatively good, while recovery in SS, RF(M), GL(M) and GL(B) was less than in other regions.

In the normalized autocorrelograms (Fig. 1C) of the above EEGs before and 20 minutes after destruction, changes due to destruction were rather obscure probably because the autocorrelograms had been normalized. However, the power spectra (Fig.1D) of these EEGs showed effects of the destruction. In the spectra of PS, not only 11−12 c/s activities, but also slow activities of 0.5−3 c/s were enhanced after destruction, whereas most activities in other regions, except the bipolar lead in GL, were depressed by destruction, though 6 c/s activities in SS were augmented slightly.

Fig. 2A shows an example of almost complete destruction of only GL. In the control EEGs before destruction (Fig.2B, left), spindle-like fast

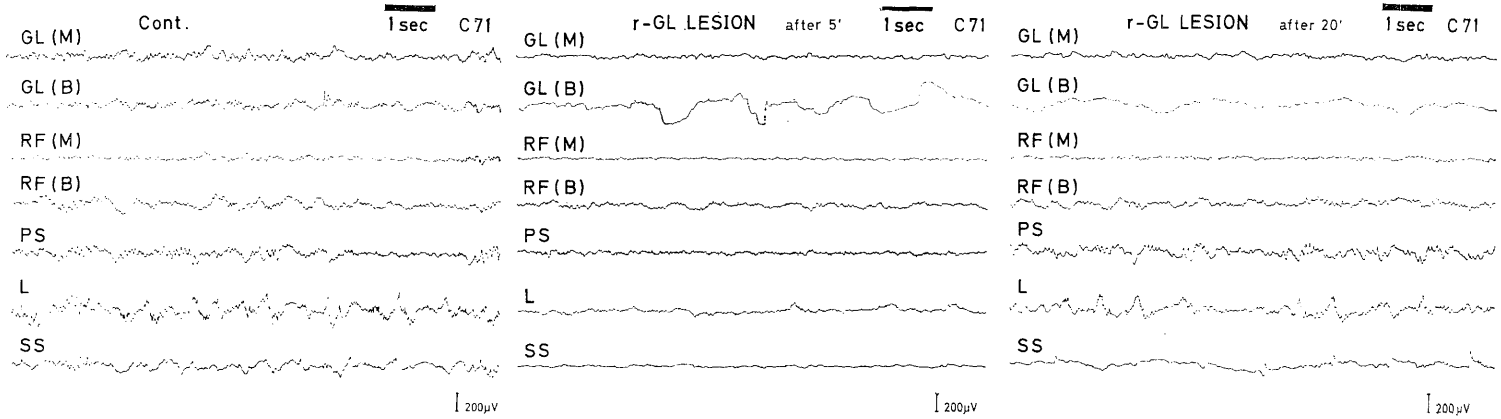
A.

Fig. 1A. Photograph of transverse section of brain (cat #71) showing destruction of dorsolateral portion (A layer) of right lateral geniculate body and neighbouring white matter.

Fig. 1B. EEG inkrecords before and after destruction demonstrated in Fig. 1A.

Left, Middle and Right: Before, 5 minutes after, and 20 minutes after destruction, respectively. GL(M) and GL(B): Mono- and bipolar leads from the lateral geniculate body, respectively. RF(M) and RF(B): Mono- and bipolar leads from the mesencephalic reticular formation, respectively. PS, L and SS: Monopolar leads from the posterior sigmoid, lateral and lateral suprasylvian gyri, respectively. All are ipsilateral records of destruction. See text.

B.



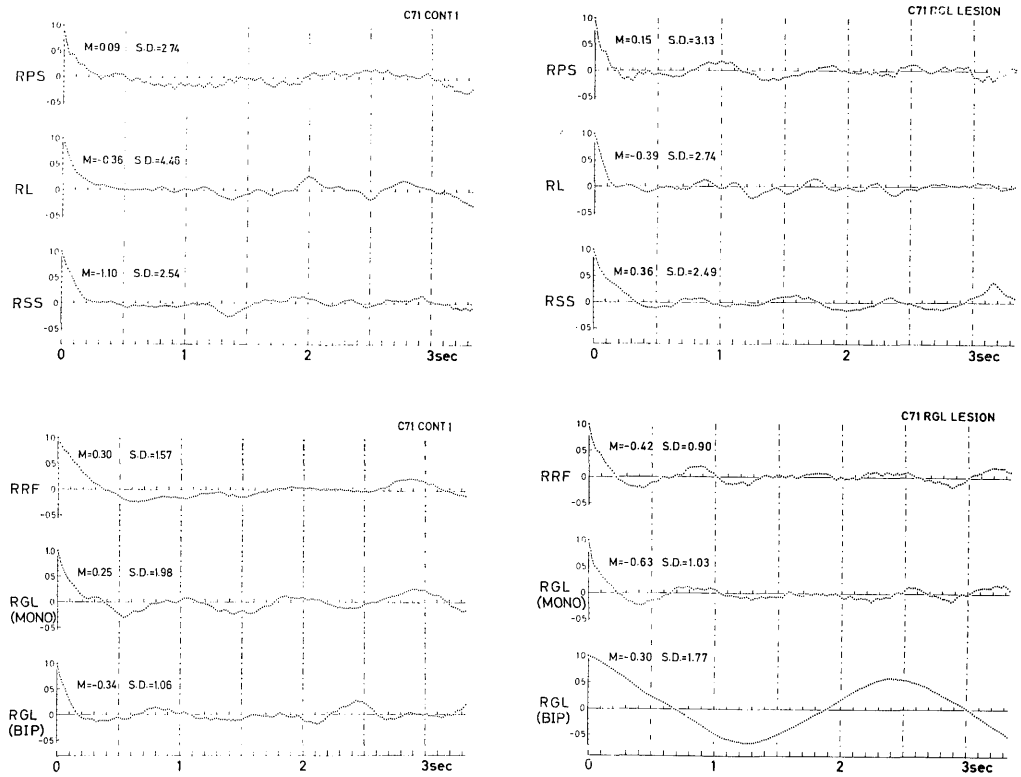


Fig. 1C. Autocorrelograms of EEGs before (left) and 20 minutes after destruction (right).

600 discrete data were sampled at 1/60 sec intervals from the EEG record shown in Fig.1B over a 10 sec length, and its autocorrelogram was computed by general purpose digital computer (HITAC 5020 or FACOM 270−20). Mean and S.D.: Mean and standard deviation of the discrete time series of EEG.

oscillations af about 24 c/s and about 12 c/s were superimposed on slow waves of about 1−1.5 c/s which suggested a stage of spindle sleep. After destruction of GL, this spindle wave of about 24 c/s was depressed (Fig. 2B, right). Relatively regular oscillations seen in the normalized autocorrelograms of EEGs before destruction changed to irregular oscillations after destruction (Fig. 2C). In addition, the power spectra (Fig .2D) depicted depression of almost all slow activities of less than 6 c/s in all regions including the L_3, L_2, SS_3, SS_2 and GL.

Even partial destruction of GL, such as demonstrated in Fig. 3A which is an example of dorsolateral lesion corresponding to the A and A_1 layers, was found to produce depression of the 8−12 c/s oscillation and disappearance of the slow wave of 0.5 c/s in the lateral (1-L) and association areas (1-SS) (Fig. 3B) of the contralateral side of GL destraction. On the side of the GL destruction, all activities in the lateral gyrus (r-L) were depressed, whereas in the posterior sigmoid gyrus (r-PS) the slow wave of about 0.5 c/s was depressed, though the fast

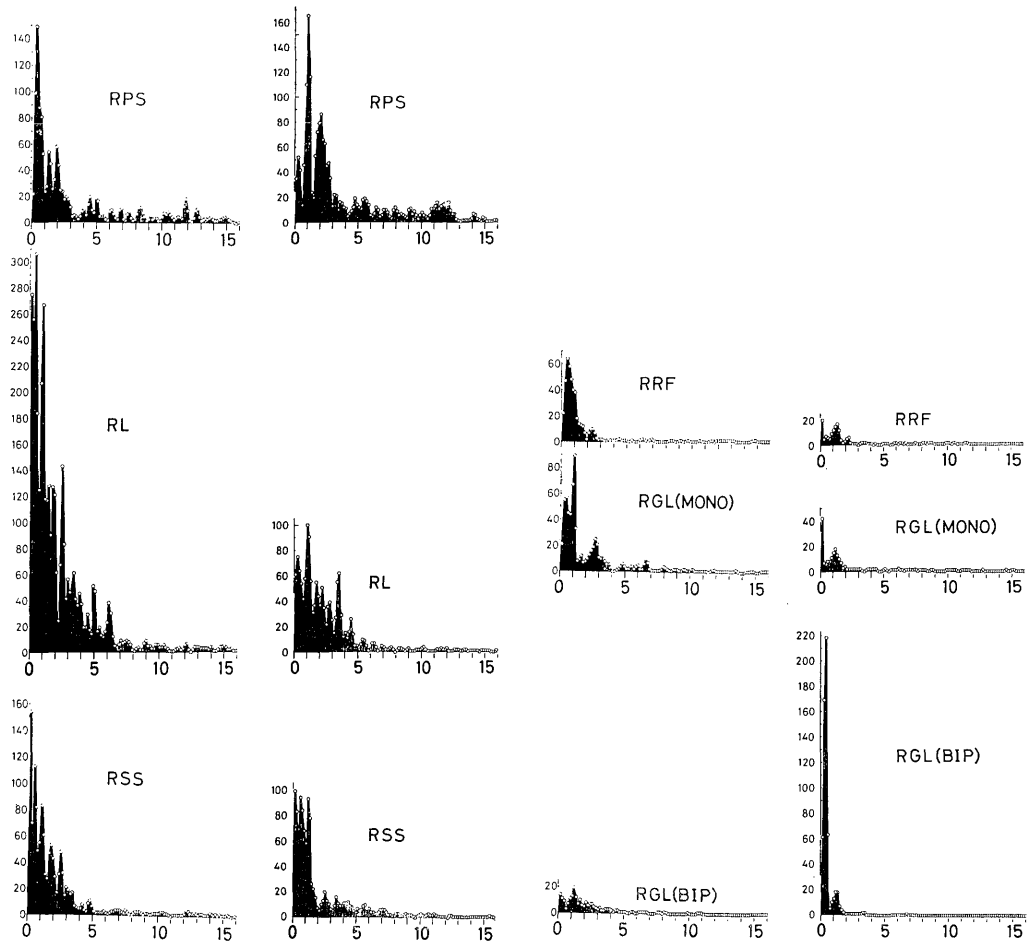
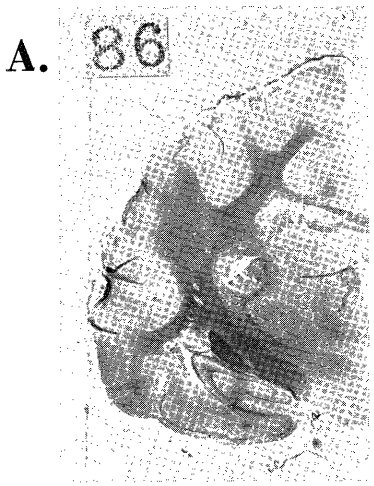


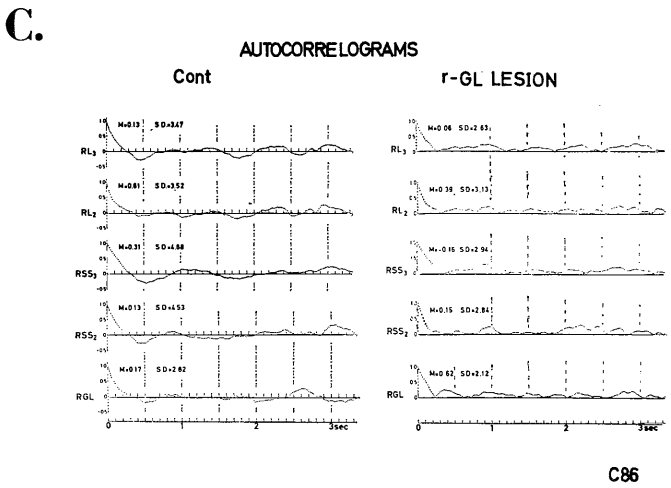
Fig. 1D. Power spectra of EEGs obtained from the autocorrelograms (Fig. 1C) through general purpose digital computer.
Abscissa: Frequency (Hertz). Left and Right: Before and 20 minutes after the destruction of right GL. Abbreviation see Fig. 1B. R indicates right side.

activity of 16 c/s in this gyrus remained the same as in Fig. 1.

In the power spectra of EEGs (Fig. 3D), the slow activities of less than 5 c/s in the ipsilateral lateral gyrus (RL) and those of less than about 3 c/s in the contralateral suprasylvian gyrus (LSS) were markedly depressed, whereas those in the lateral gyrus (LL) contralateral to the GL destruction were only slightly depressed, and activities of about 1 c/s were enhanced in the contralateral optic radiation (LOR). Depressive effects of destruction were also suggested on the normalized autocorrelograms (Fig. 3C) and by the standard deviations (Fig. 3C and Table 1) of EEGs before and after destruction.

As demonstrated in Fig. 1,2 and 3, some EEG activities of less than 5 c/s, such as 0.5, 1, 1.5 c/s etc. in lateral (visual), suprasylvian (association) gyri were depressed not only on the same side but also


A.
86


C.
AUTOCORRELOGRAMS
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RL2
RSS3
RSS2
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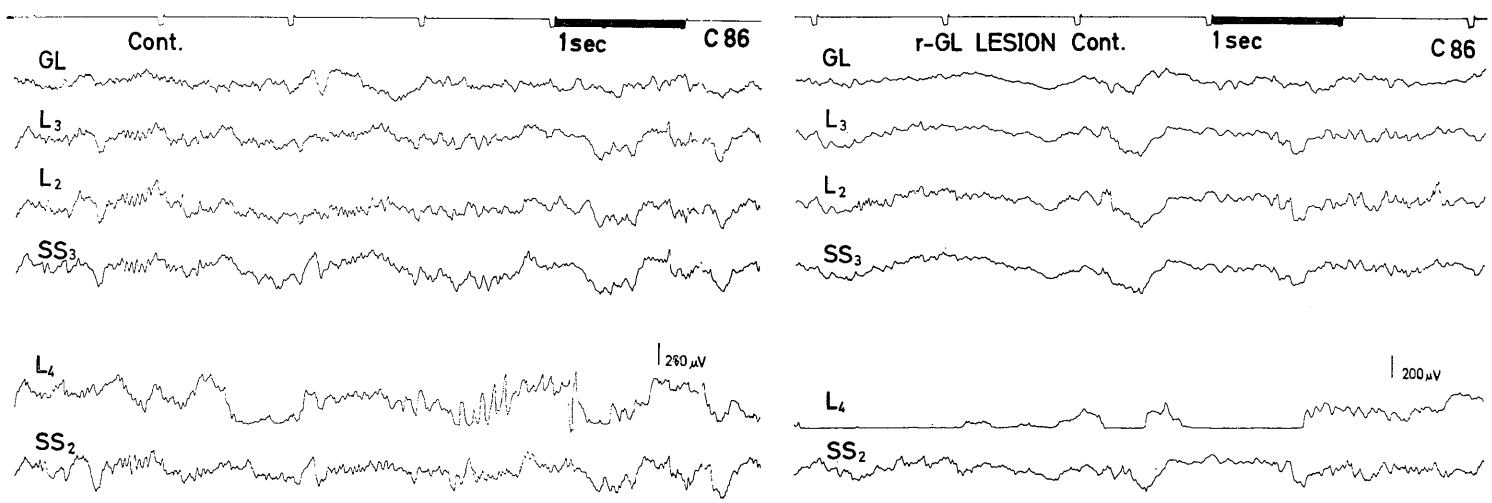
B.
Cont.
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SS3
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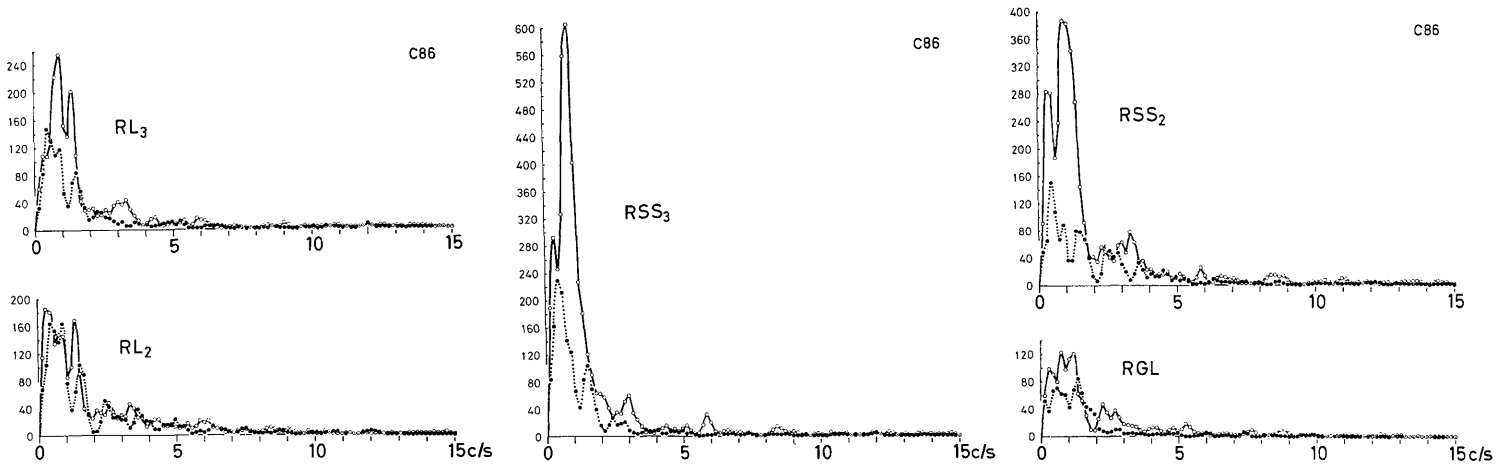


Fig. 2A. Photograph of transverse section of brain with almost complete destruction of lateral geniculate (cat #86).

Fig. 2B. EEG records before (left) and twenty minutes after (right) destruction of right lateral geniculate body. L_4, L_3 and L_2: lateral gyrus. SS_3 and SS_2: lateral suprasylvian gyrus. See Fig. 6.

Fig. 2C. Autocorrelograms of EEG records before (left) and 20 minutes after destruction of the right lateral geniculate body (RGL).

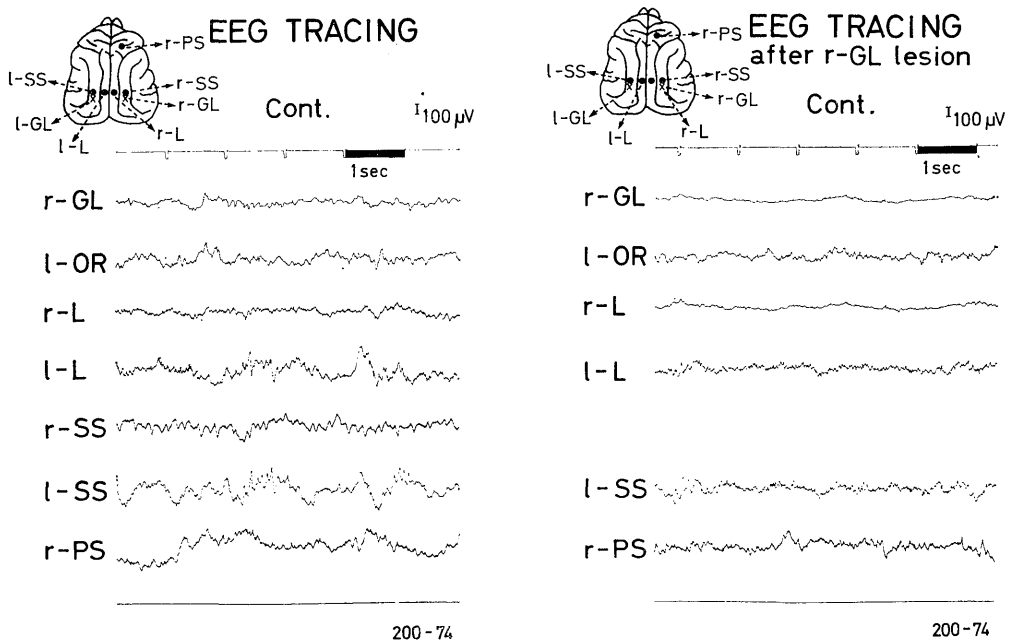
Fig. 2D. Power spectra of the autocorrelograms in Fig. 2C.
White circles and bold line: Before destruction of right lateral genicalate body. Black circles and dotted line: 20 minutes after destruction. Explanation, see text.

A.

Fig. 3A. Photograph of transverse section of brain (cat #74) wth partial destruction of right lateral geniculate body (A and A_1 layer) and neighbouring white matter.

Fig. 3B. EEG records before (left) and 20 minutes after destruction (right) shown in Fig. 3A.
OR: Optic radiation. L: Lateral gyrus L_3.
SS: Lateral suprasylvian gyrus SS_3. See text.

B.



on the opposite side of the destruction of GL and/or the cerebral white matter, whereas EEG activities in the posterior sigmoid (somatosensory) gyrus (PS) were not supressed but enhanced.

The three examples demonstrated thus far manifested mainly depression of a variety of EEG activities caused by destruction of unilateral GL. The example illustrated in Fig.4, on the contrary, showed some augmentations by destruction of the dorsolateral (A layer) portion of

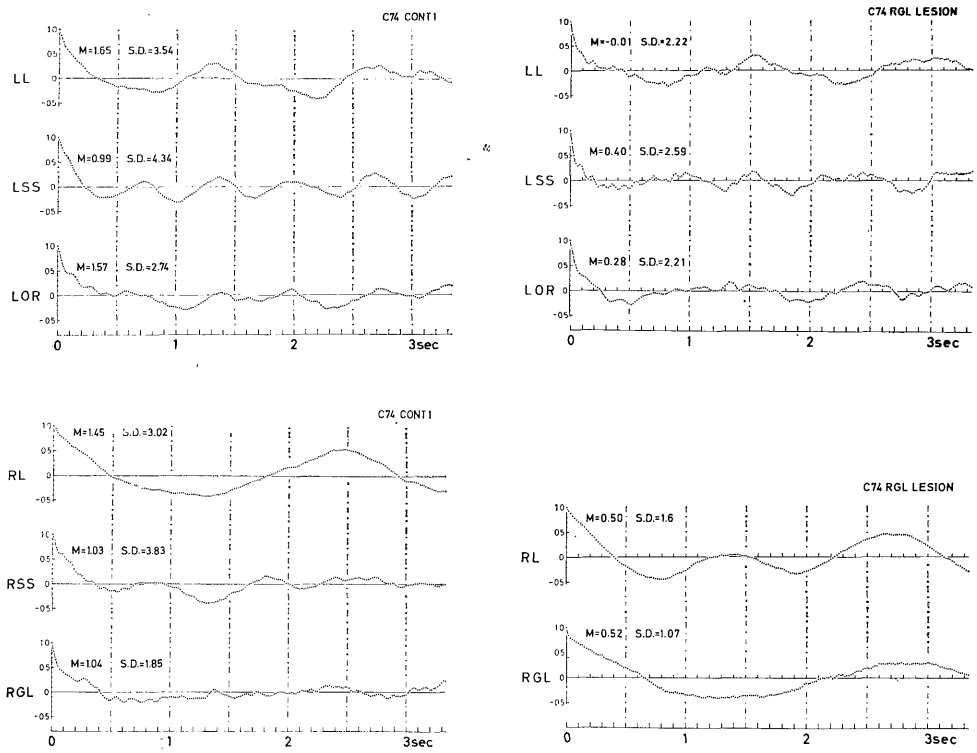


Fig. 3C. Autocorrelograms of EEGs before (left) and twenty minutes after GL destruction (right).

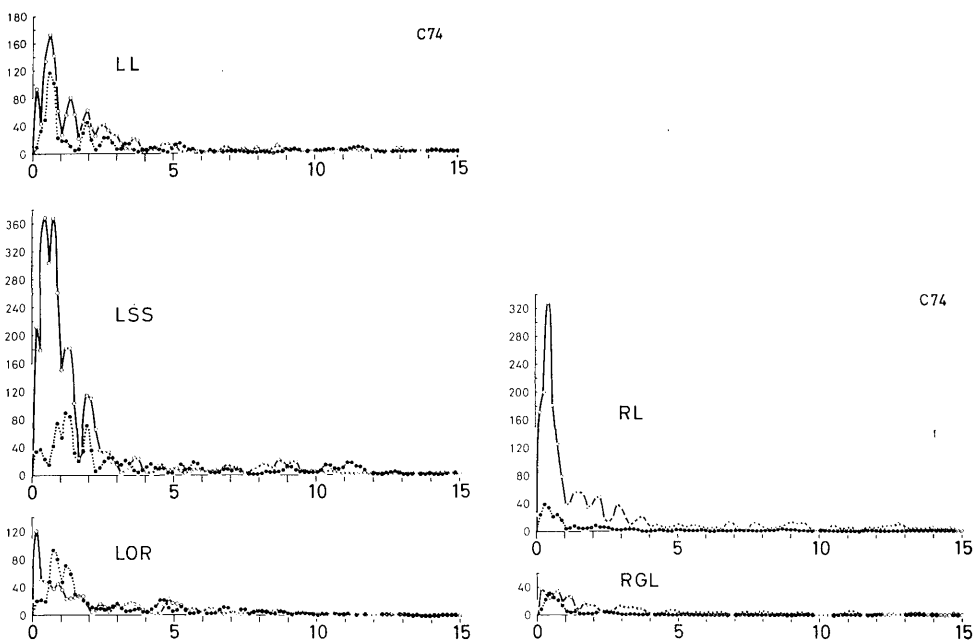


Fig. 3D. Power spectra of the autocorrelograms in Fig. 3C. See text.

		RPS	RL	RSS	RRF	RGL(M)
C−71	Before	2.74	4.46	2.54	1.57	1.98
	After	3.13*	2.74	2.49	0.90	1.03
		RL	RSS	LL	LSS	LOR	RGL
C−74	Before	3.20	3.83	3.54	4.34	2.74	1.85
	After	1.60	−	2.22	2.59	2.21	1.07
		RL	RSS	LL	RRF	RGL	RCP
C−88	Before	3.42	3.85	5.09	1.73	2.94	2.23
	After	3.20	3.54	5.75*	1.56	1.79	1.91
		RL_3	RL_2	RSS_3	RSS_2	RGL
C−86	Before	3.47	3.52	4.68	4.53	2.82
	After	2.63	3.13	2.94	2.84	2.12

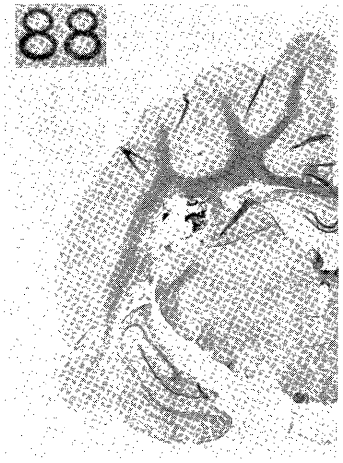
Table 1. Standard deviations of background EEGs in various regions before and after destruction of a lateral geniculate body.

600 ordinates of an EEG were sampled at 1/60 sec intervals over a length of 10 sec, by which an autocorrelogram was computed, so that square of the standard deviation is the ordinate of the autocorrelogram at the time origin. All differences in standard deviations before and after the destruction were statistically significant (F-test, P=0.01). It was suggested, therefore, that most EEGs had been depressed by destruction, whereas that in the posterior sigmoid gyrus (PS) was augmented.

the unilateral GL and partial destruction of the cerebral white matter (Fig. 4A) around the depth electrode. In this instance, in spite of considerable depression of almost all activities of various frequencies in the subcortical regions (r-GL, r-CP, and r-RF in Fig. 4B, C and D), some accelerative changes were verified in the cortical EEG activities, such as the 3.5−4.5 c/s activities in RL, LL and RSS, the approximately 1 c/s activity in RL and RSS, and the 6−9 c/s activities in LL (Fig. 4D). However, considerable depression was seen in the 0.5 c/s activities in the three cerebral regions (RL, LL and RSS) and in the approximately 10 c/s activities in the lateral (RL) and suprasylvian gyri (RSS) on the side of destruction.

The above findings show that destruction of a lateral geniculate body (GL) mainly causes depression of EEG activities in the lateral, suprasylvian gyri and some subcortical sites.

To determine whether these changes are specific to GL destruction or not, destruction of another structure by the same condition as used in GL destruction was performed in two cats. Fig. 5A shows the results caused by putamen (A:12.0, L:10.5, H:−1.0) destruction. In this instance, no depressive activities were observed in the EEG ink records in various cortical and subcortical sites after this procedure, as depicted

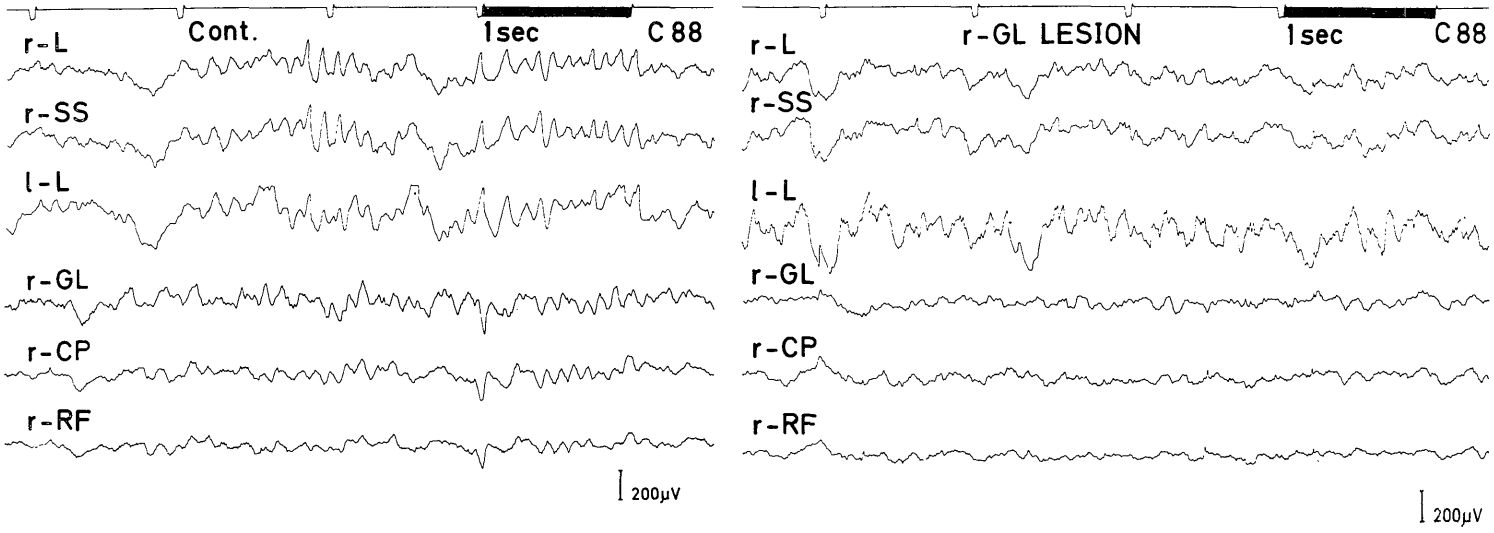


A.

Fig. 4A. Photograph of transverse section of brain (cat #88) showing destruction of right lateral geniculate body (A layer) and neighbouring white matter.

Fig. 4B. EEG inkrecords before (left) and twenty minutes after destruction (right). CP: Commisula posterior. See text.

B.



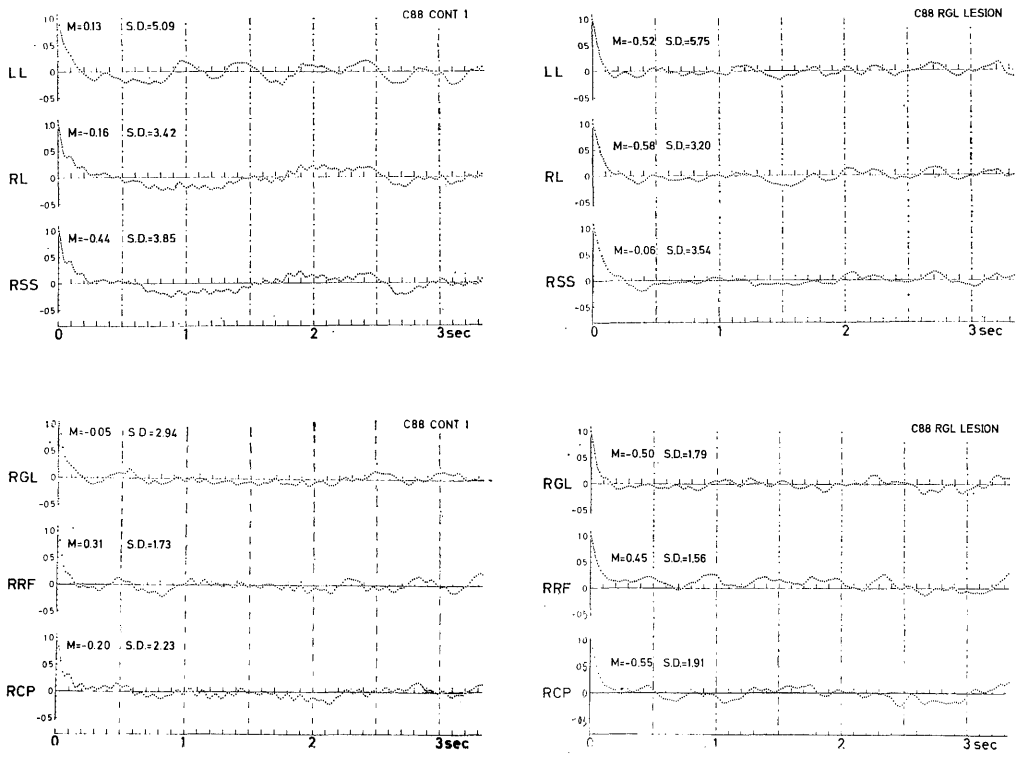


Fig. 4C. Autocorrelograms of EEGs before (left) and after destruction (right). LL and RL: Left and right lateral gyri, respectively. See text.

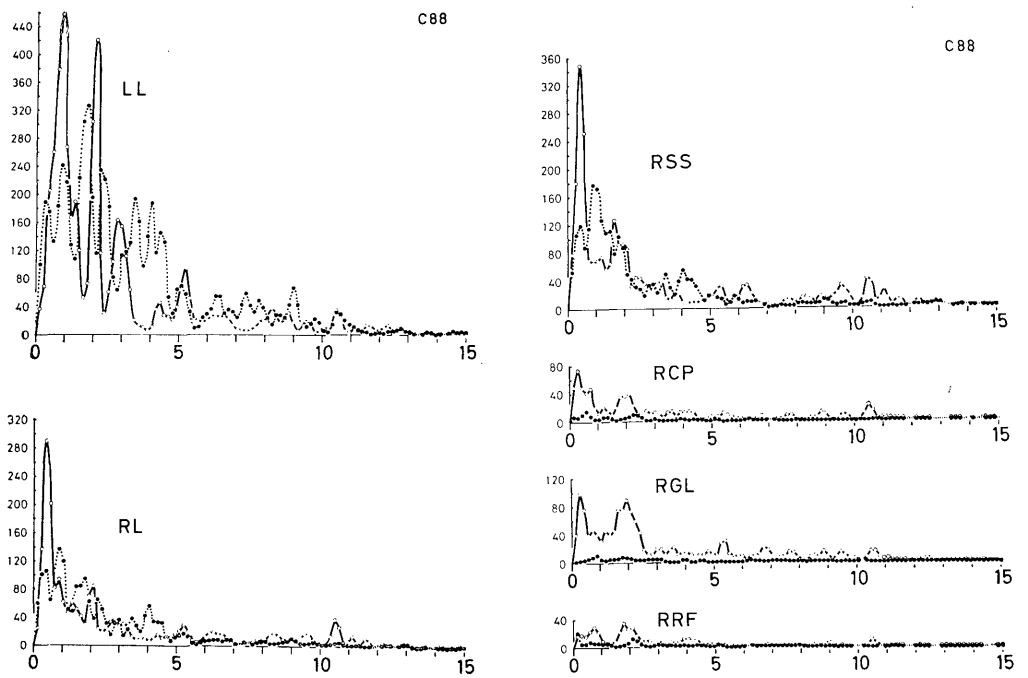
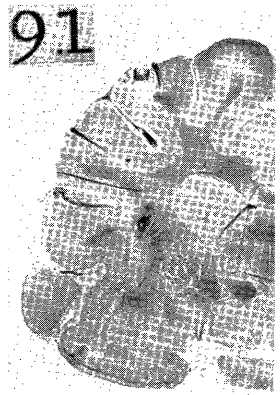


Fig. 4D. Power spectra of EEG. Abbreviation, see Fig. 4B.

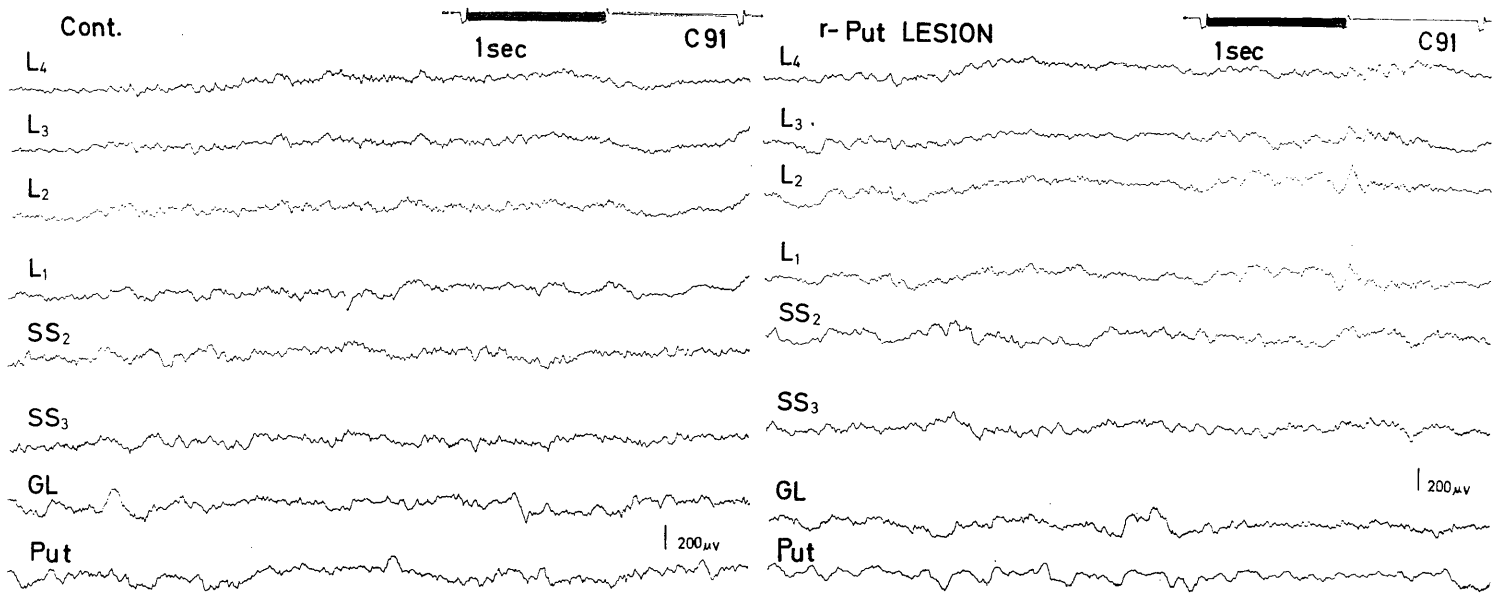


A.

Fig. 5A. Photograph of transverse section of the brain (cat # 91) showing right putamen destruction.

Fig. 5B. EEG inkrecords before (left) and twenty minutes after destruction (right). Put: Putamen. See text.

B.



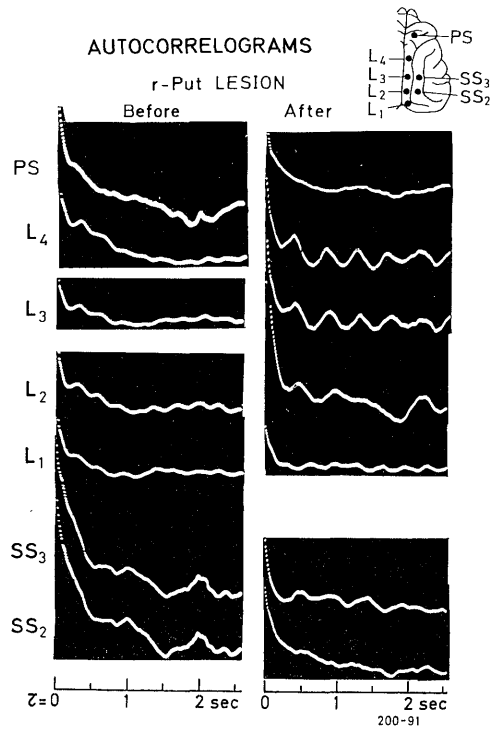


Fig. 5C. Autocorrelograms of EEGs.

Autocorrelograms were computed by ATAC−501−20 and magnetic tape records of EEGs of 26.2 sec length (analysis time). Total delay (length of each autocorrelogram) is 2.621 sec. Left and Right: Before and twenty minutes after right putamen destruction.

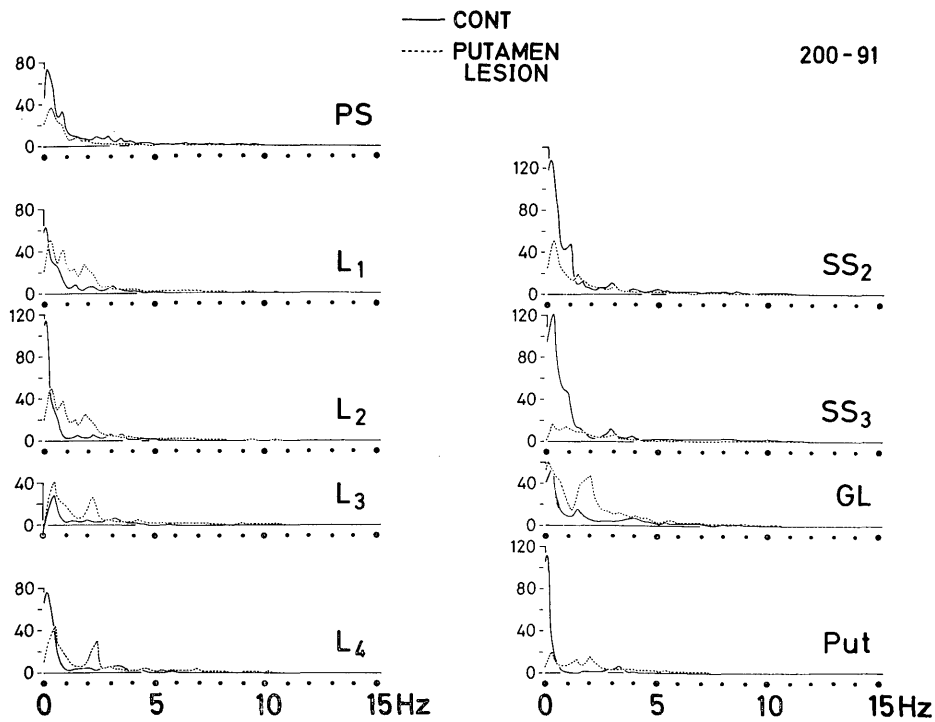


Fig. 5D. Power spectra of EEGs.

Each autocorrelogram obtained by ATAC−501−20 was printed out in a digital time series of 256 data through the Digital Printer (ATAC 120 and ATAC 125) at every 10.2 msec.

in Fig. 5B, but rather there was enhancement of some activities.

It was observed that oscillations of about 2 c/s in the autocorrelograms of EEGs led from various regions in the lateral gyrus (L_2,L_3,L_4) (Fig.5C) had been augmented after destruction. In addition, the frequency spectra (Fig. 5D) of the EEGs, which had been obtained by the FACOM computer from the digital time series of the computed autocorrelogram (Fig. 5C) by ATAC-501−20 through the Digital Recorder (ATAC 120 and ATAC 125), showed that not only the peaks of about 2 c/s activities, but also peaks of slower frequencies of about 1c/s,etc. in the lateral gyrus, lateral geniculate body and putamen were enhanced after putamen destruction. On the contrary, peaks in the postsigmoid (PS) and lateral suprasylvian gyri (SS_2, SS_3) were depressed after destruction. Almost all of these findings were not only different from those observed by destruction of the lateral geniculate body, but even contradictory.

II. *Influences of GL lesion on the averaged evoked potentials.*

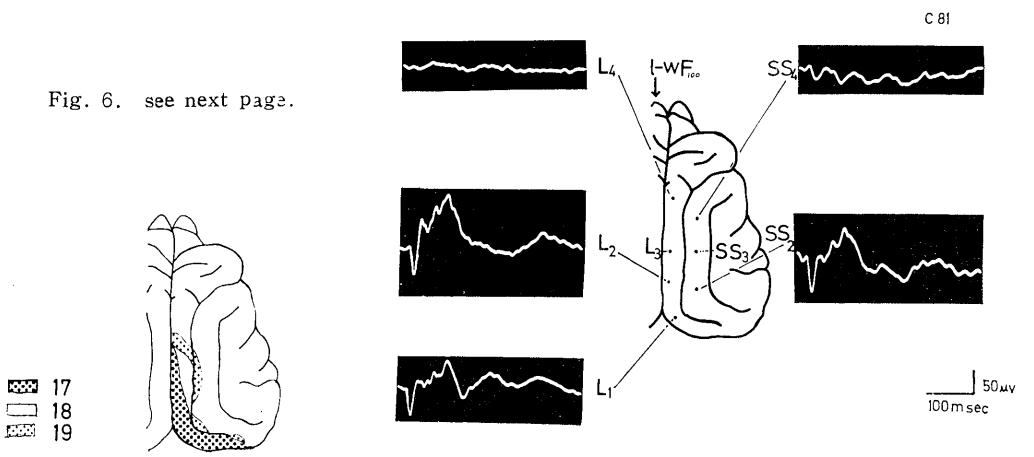
1). Averaged VEP in the visual and association areas.

By referring to the cortical surface extent of anatomical areas (Otsuka and Hassler 1962[16]), electrodes at L_1,L_2 and L_3 in the lateral gyrus were located in the primary visual area 17, and electrodes at L_4 were in the anterior lateral gyrus, while those at SS_4,SS_3,SS_2 in the lateral suprasylvian gyrus were in the association area (Fig.6).

Averaged VEPs to flashing light stimuli obtained by summation technique at L_1,L_2,L_3,SS_2 and SS_3 manifested an initial positive-negative diphasic deflection followed by some positive-negative potentials, whereas VEPs led from the electrodes at L_4 and SS_4 did not demonstrate this initial deflection, as illustrated in, Fig. 6.

The onset latency of the initial positive response brought out in the lateral suprasylvian gyrus (association area) SS_3 was the same at that obtained in the primary visual area L_3, which was 32

Fig. 6. see next page.



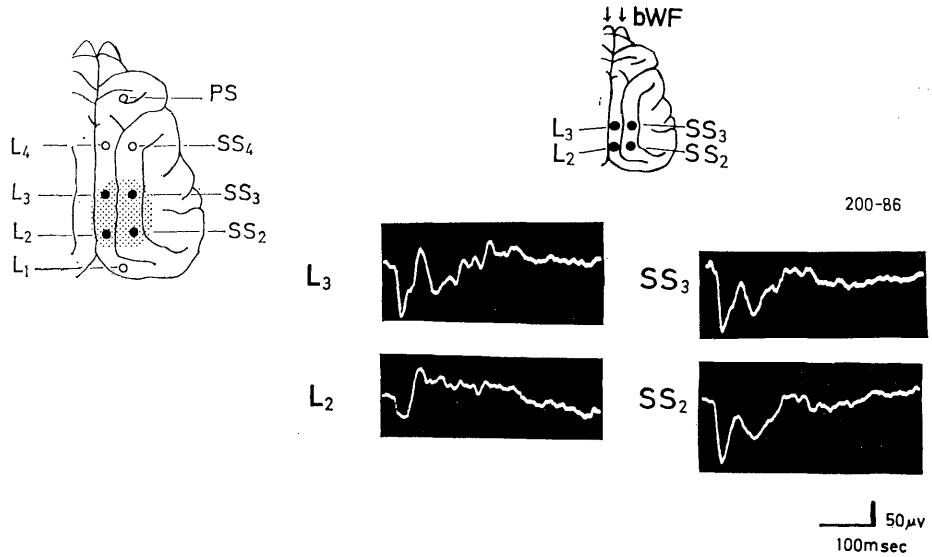


Fig. 6. Visually evoked potentials in the cerebral cortex.

Left: Cerebral primary (17) and secondary (18, 19) visual areas (Top) and locations of monopolar leads (Bottom) in the postsigmoid (PS), lateral (L_4, L_3, L_2 and L_1), and lateral suprasylvian gyri (SS_4, SS_3, SS_2), respectively.

Right: Visually evoked potentials (VEPs) elicited by a contra-lateral monocular flash stimulus (Top) and binocular flash stimulus (Bottom).

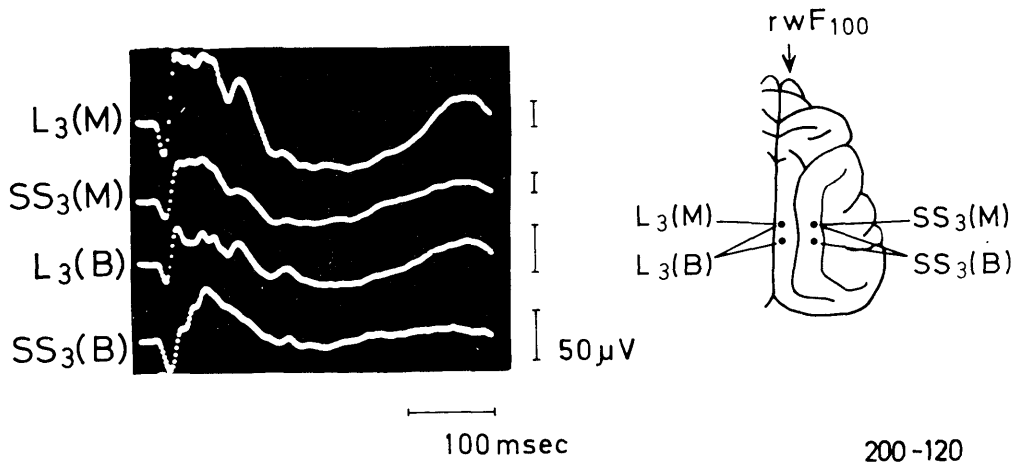


Fig. 7. Visually evoked potentials in the lateral (L_3) and lateral suprasylvian gyri (SS_3) led mono- and bipolarily.

Bipolar electrodes were placed 3 mm apart. Difference in the initial positive deflection between the lateral (L_3) and suprasylvian gyri (SS_3) were more prominent in bipolar records than those in monopolar records.

msec in the instance shown in Fig. 7. However, in comparison with those led by bipolar leads, the peak latency and duration of the former response were noted to be longer than those of the later, being 35 msec and 30 msec, respectively.

Each of the initial and late positive-negative deflections obtained by summation of thirty responses through the computer (ATAC-501−20) from VEPs due to unilateral stimulus were labelled P_1, N_1, P_2, N_2, P_3, N_3, ··· in the order of appearance after the method of HIROTA (1965)[9]. It was found that the initial and fifth surface positive deflections, P_1 and P_5, correspond to wave 1 and 8, while the first and fourth negative deflections, N_1 and N_4, coincide with waves *a* and *g* described by STEINBERG (1965)[18]. Moreover, P_1, N_1, N_4 correspond to waves 1°, 1, ··· ,7 of NORTON and JEWETT (1967)[14]. The onset latency of the P_1 deflections are illustrated in Table 2.

(2) Peak latency (msec)

		On-L	P_1	N_1	P_2	N_2	P_3	N_3	P_4	N_4	P_5	N_5	P_6	N_6
	C-71	20	30	40	55	60	—	—	95	110	120	130	160	185
	C-74	20	32	45	—	55	60	78	92	104	115	125	145	-
	C-75	20	30	40	50	60	65	78	95	108	115	125	160	—
	C-78	18	25	38	50	60	75	86	92	110	125	140	160	190
	C-80	20	34	45	50	55	68	85	90	95	110	115	—	—
L_3	C-82	20	35	45	50	60	—	—	—	—	—	—	—	—
	C-83	20	32	50	55	65	75	80	95	105	120	130	140	150
	C-86	20	35	50	57	70	75	85	100	117	125	145	157	165
	C-87	20	30	42	47	57	68	75	90	105	—	—	—	—
	C-88	20	38	55	58	—	—	80	92	100	120	130	—	—
	C-91	20	30	52	65	68	72	80	85	90	105	120	—	—
	AVE	19.8	31.9	45.6	53.7	61.0	69.8	80.7	92.6	104.4	117.2	128.8	153.7	172.5
	(±)$\triangle_{0.05}$	0.3	2.1	3.4	6.6	3.7	4.7	3.0	2.9	5.7	5.2	7.2	8.7	30.1
	C-79	20	35	50	—	—	75	90	—	100	—	—	—	—
	C-80	20	35	50	—	55	75	80	90	100	—	—	—	—
	C-81	15	30	40	55	75	—	—	80	110	125	—	—	—
L_2	C-84	20	35	45	55	70	—	—	95	105	—	—	—	—
	C-86	20	30	50	55	70	75	85	100	115	125	135	—	—
	C-91	20	30	50	65	70	80	90	95	100	110	125	-	—
	AVE	19.2	32.5	47.5	57.5	68.0	76.2	86.2	92.0	105.0	120.0	130.0		
	(±)$\triangle_{0.05}$	2.2	2.8	4.4	14.6	9.4	4.1	8.9	9.4	6.7	21.9			
	C-79	20	35	50	63	68	75	—	—	—	—	—	—	—
	C-81	20	30	43	55	65	68	75	85	100	—	—	—	—
	C-93	20	33	50	53	70	—	—	88	—	—	—	—	—
L_1	C-84	15	28	53	—	—	—	—	—	—	—	—	—	—
	C-85	23	32	45	57	68	—	-	—	—	—	—	—	—
	C-87	15	28	40	45	50	63	70	83	95	—	—	—	—
	C-91	20	32	52	—	—	—	—	84	90	95	100	—	—
	AVE	19.0	31.1	47.6	54.6	65.2	68.7	72.5	85.0	95.0				
	(±)$\triangle_{0.05}$	2.8	5.9	4.6	7.8	7.2	12.6		3.51	3.5	12.5			

		On-L	P_1	N_1	P_2	N_2	P_3	N_3	P_4	N_4	P_5	N_5	P_6	N_6
SS_3	C-71	15	35	45	—	—	75	—	—	105	—	—	—	—
	C-76	20	32	50	65	80	90	100	—	—	—	—	—	—
	C-73	20	40	—	—	80	—	—	—	—	—	—	—	—
	C-78	20	32	45	65	—	—	100	—	—	—	—	—	—
	C-79	20	35	55	—	—	75	—	--	108	—	—	—	—
	C-86	15	35	50	60	70	76	85	100	120	125	145	—	—
	C-88	20	35	62	65	75	80	100	—	—	125	140	—	—
	C-91	20	30	45	60	70	82	90	105	117	—	—	—	—
	AVE	18.8	34.2	50.2	63.0	75.0	79.6	95.0		112.5	125.0	142.5		
	(±)△0.05	2.0	2.5	6.0	3.3	6.2	5.8	6.3		10.2				
SS_2	C-80	20	35	45	—	—	75	80	90	100	—	—	—	—
	C-81	20	30	40	50	55	60	80	95	110	—	—	—	—
	C-83	20	35	50	55	70	—	—	95	100	115	135	—	—
	C-84	20	30	45	55	70	—	—	95	105	—	—	—	—
	C-86	20	35	55	60	75	80	85	105	120	130	150	—	—
	AVE	20.0	33.0	47.0	55.0	67.5	71.7	81.7	96.0	107.0	122.5	142.5		
	(±)△0.05		3.3	7.2	6.4	14.0	26.2	7.3	6.8	10.4				

(2) Peak to peak amplitude (μV)

		$\overline{P_1}$	$\overline{P_1N_1}$	$\overline{P_2}$	$\overline{P_2N_2}$	$\overline{P_3}$	$\overline{P_3N_3}$	$\overline{P_4}$	$\overline{P_4N_4}$	$\overline{P_5}$	$\overline{P_5N_5}$	$\overline{P_6}$	$\overline{P_6N_6}$
L_3	C-71	300	212	100	112	62	—	50	50	75	25	200	100
	C-74	68	156	—	43	31	37	63	25	25	6	94	—
	C-75	83	133	78	50	11	11	100	61	38	16	38	—
	C-78	80	115	45	10	45	10	35	35	50	35	10	45
	C-80	55	45	10	10	15	30	15	15	20	5	—	—
	C-82	17	83	5	22	—	22	—	—	—	—	—	—
	C-83	56	175	25	50	12	10	43	50	31	50	18	18
	C-86	62	112	25	56	25	12	118	75	37	50	37	25
	C-87	56	81	12	50	43	—	25	18	—	—	—	—
	C-88	50	125	—	—	—	87	21	18	75	31	—	—
	C-91	44	133	50	6	17	15	15	16	16	22	—	—
	AVE	79.2	124.5	39.0	40.9	29.0	26.0	48.5	36.3	40.7	26.7	66.1	47.0
	(±)△0.05	46.5	28.6	25.2	25.9	13.7	19.2	26.9	15.9	16.9	13.7	71.2	60.4
L_1	C-79	6	50	18	12	12	—	—	—	—	—	—	—
	C-81	70	115	40	40	20	30	15	40	—	—	—	—
	C-83	25	62	6	18	—	—	18	—	—	—	—	—
	C-84	36	50	—	—	—	—	—	—	—	—	—	—
	C-85	21	50	14	14	—	—	—	—	—	—	—	—
	C-87	68	75	6	38	55	25	13	13	—	—	—	—
	C-91	93	137	—	—	—	—	25	43	13	20	—	—
	AVE	45.5	77.0	16.8	24.4	29.0	27.5	17.7	32.0				

	(±)$\triangle_{0.05}$	25.3	30.9	17.4	17.9	39.9		8.4	41.5			
SS_3	C-71	17	28	—	—	50	—	—	56	—	—	—	—
	C-73	12	—	—	44	—	—	—	—	—	—	—	—
	C-76	17	25	25	17	25	—	—	—	—	—	—	—
	C-78	10	20	20	—	—	15	—	—	—	—	—	—
	C-79	28	111	—	—	56	—	—	100	—	—	—	—
	C-86	94	94	25	31	6	6	94	63	13	63	—	—
	C-88	50	119	6	25	6	31	—	—	68	31	—	—
	C-91	63	100	25	—	56	25	18	18	—	—	—	—
	AVE	36.3	71.0	12.5	29.2	32.5	31.2	56.0	59.2				
	(±)$\triangle_{0.05}$	25.5	37.7	14.9	56.6	23.8	12.5		54.5				

Table 2. Onset and peak latencies and peak to peak amplitudes of each response component in the visually evoked potential elicited by contralateral flash in the lateral (L_3, L_2, L_1) and lateral suprasylvian gyri (SS_3, SS_2).

(1) Onset and peak latencies,(msec)
On-L:Onset latency.
AVE: Average
$\triangle_{0.05}$: Fiducial interval under 0.95.

(2) Peak to peak amplitude (μV)

Within 150 msec after flashing light stimulus, five diphasic deflections time-locked to the stimulus were observed. In the VEP led from the caudal portion of the lateral gyrus (L_1) of seven cats, response P_2 and later responses seemed to be smaller in size (Table 2) than those led from the anterior and midlateral gyrus, L_3 and L_2. Hardly any of the cats produced P_5 and N_5. On the other hand, VEPs in the lateral suprasylvian gyrus SS_3, and SS_2, association area, manifested five diphasic deflections.

Peak-to-peak amplitudes, which were classified into five groups, and the onset and peak latencies of every deflection of VEP in each animal area are depicted in Fig.8. It was shown that each response component of the VEP in L_1 is smaller in amplitude than those in other regions (visual area L_3, L_2 and association areas SS_3, SS_2). P_1 and N_1 were stable in their peak latencies with individual variation of 10−15 msec, and the peak-to-peak amplitudes $\overline{P_1 N_1}$ were highest followed by the amplitude of the initial positive deflection P_1. Individual variations of the peak latencies of P_2 and those of later response components were large, being more than 20 msec, which suggests that they are less rigidly time locked to the flash stimulus.

Though there were differences between animals, the peak-to-peak

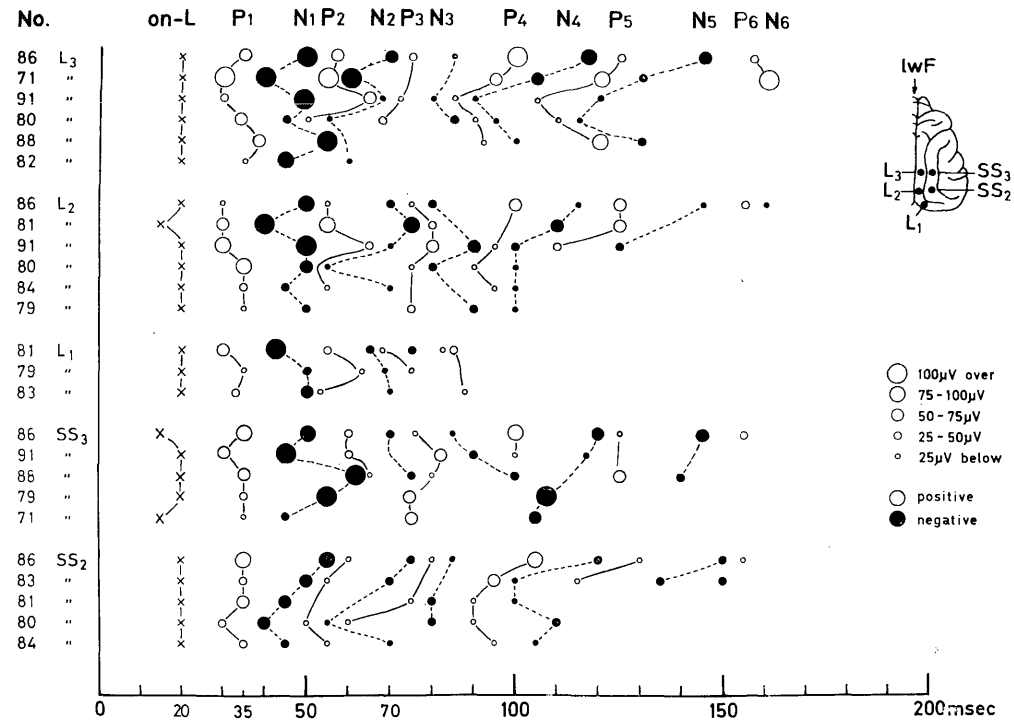


Fig. 8. Peak latencies and amplitudes of each response component in the visually evoked potential of lateral gyrus and lateral suprasylyian gyrus.

Black and white circles: negative and positive deflections, respectively. Amplitude of each component is classified into five classes: over 100, 75—100, 50—75, 25—50, and below 25μV, respectively. The higher the potential, the larger is the circle.

amplitudes of all response components and the average schematic wave form in the mid-lateral gyrusL_3, posterior lateral gyrus L_1, and lateral suprasylvian gyrus, SS_3, are illustrated in Fig. 9, wherein the averages for L_1, L_3 and SS_3 were obtained, respectively, from seven, eleven and eight cats. Comparison of the representaive wave form of VEP obtained from SS_3 with that from L_3 shown in Table 2 and Fig.9, verified that the mean peak latencies of all deflections observed in SS_3, such as P_1, N_1, P_2, N_2, P_3, N_3, P_4 and N_4, were longer than the corresponding ones in L_3. However, statistically significant differences were found only in the peak latencies of P_2, N_2 and N_3 deflections. One reason may have been the small number of VEP sampled to obtain the average because of which the fiducial intervals of the latencies were too broad to verify significant differences. Average amplitudes of deflections of the initial positive $\overline{P_1}$, $\overline{P_1N_1}$ and $\overline{P_2}$ in SS_3 seemed to be considerably lower than those in L_3, whereas those of $\overline{P_3}$, $\overline{P_3N_3}$, $\overline{P_4}$ and $\overline{P_4N_4}$, etc. in SS_3 were considerably higher than those in L_3, although not sigificant statistically.

In Fig. 10, the configurations of VEPs until 1000 msec after the

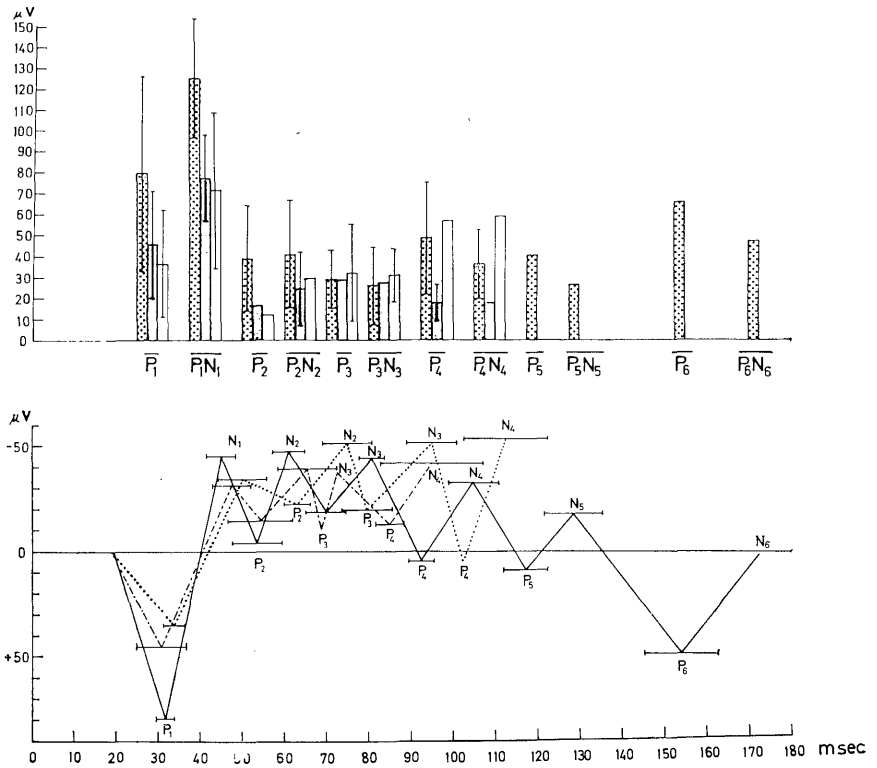


Fig. 9. Peak-to-peak amplitudes of response components and average wave forms of visually evoked potentials in the lateral and lateral suprasylvian gyri.

Top: Peak-to-peak amplitude. Dotted (left), shaded (middle) and clear (right) bars correspond to components L_3, L_1 and SS_3, respectively.

Bottom: Average wave form. Bold line; L_3. Broken line; L_1. Dotted line; SS_3.

Vertical and horizontal lines indicate variation in peak-to-peak amplitudes and peak latencies, respectively, at fiducial interval of 0.95. See text.

flash stimulus are depicted for six cats. All deflections within 130 msec after the stimulus were similar in latencies among the animals. Thereafter, slow positive (Fig. 10, C71, 74 and 75) and negative waves with peak latencies of about 150 and 200 msec were raised in some animals (Fig. 10, C74 and 75). This later diphasic wave was followed by some slow waves, which varied between animals. Although considerable variation was observed in the time configuration of VEPs, they were classified in into the following four types (Fig. 11). Type I: the initial and next diphasic deflections, P_1N_1 and P_2N_2, are considerably high, while the following diphasic deflections P_3N_3, P_4N_4, etc. are not so high. Type II: the initial diphasic deflection P_1N_1 is followed by a prominent slow negative swell of about 70 msec duration, onto which P_2N_2, P_3N_3 are superimposed, and the succeeding positive trough P_4 shows a considerably marked swing so that $\overline{N_3P_4}$ is prominent. Type III: the primary positive response P_1 is relatively low, and N_1, P_2N_2, P_3N_3, etc. are superimposed on a slow negative wave of about 150

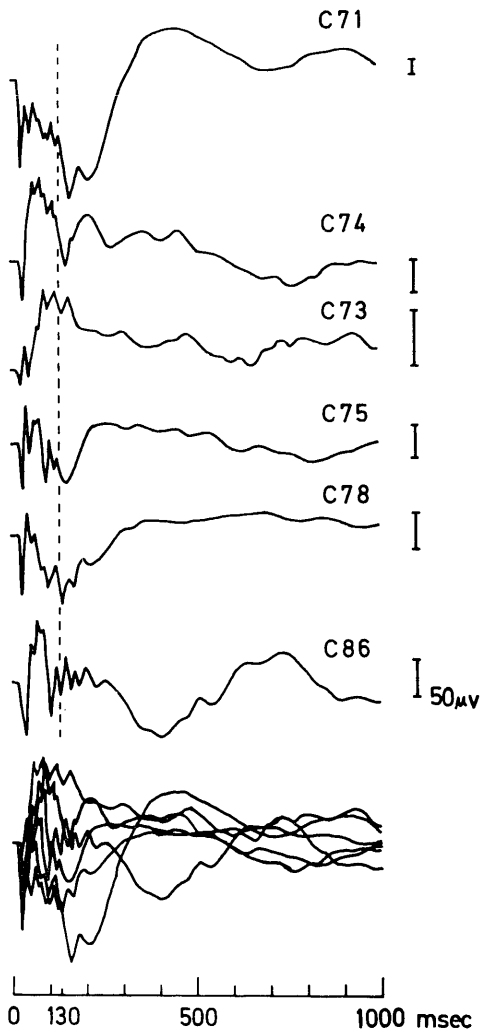


Fig 10. VEPs over a length of 1000 msec after contralateral monocular flash stimulus obtained from the primary visual area (L_3).
Each VEP was obtained by thirty times summation. Bottom: Superimposition of six VEPs depicted in the above.

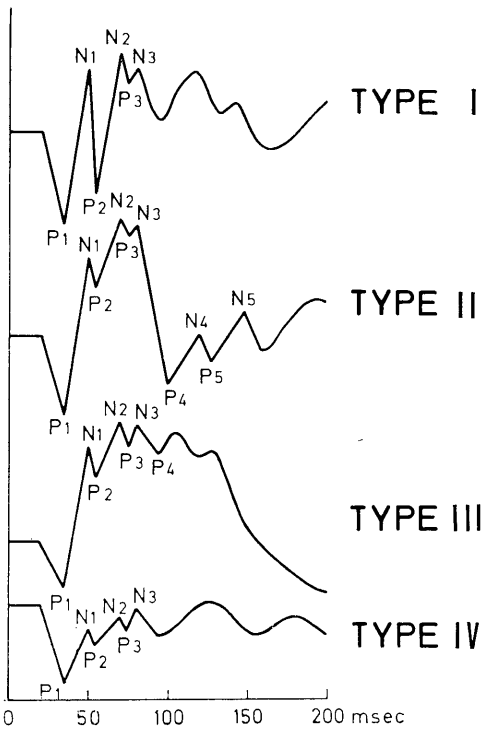


Fig. 11. Types of wave forms in VEP of 200 msec obtained from the primary visual area (L_3).

msec duration. Type IV: all diphasic deflections P_1N_1, P_2N_2, P_3N_3, etc. are superimposed on a slow positive downward wave.

2). Effects of GL destruction on VEP.

In case of almost destruction of GL (Fig. 2A), not only was the inital positive-negative response P_1N_1 in the lateral and lateral suprasylvian gyri (L_3 and SS_2 in Fig. 12) wiped out, but the later diphasic responses P_2N_2, ···, P_5N_5 were also almost completely deteriorated after destruction. In place of these missing responses, a slow negative

response of 70 msec peak latency appeared lasting for 120 msec. In contrast to this, when, in stead of complete GL destruction, only partial damage had been inflicted upon the dorsolateral portion of GL (Fig. 4A), no change was observed in the initial positive response P_1, but almost all of the later responses were depressed in the VEP elicited by ipsi-or contralateral monocular flashing light stimulus (Fig.13A). However, the depression was milder in the VEP brought out by binocular stimulus. In addition, the response components that are brought out after about 150 msec following the stimulus were almost all wiped out when the destruction of GL was considerable, as demonstrated in Fig.13B. The background EEGs activities in such cases were depressed promin ently by the destruction (Fig. 1 and 3). When the destruction had been produced in the ventrolateral GL and in one part of the optic tract (OT) (Fig. 14A), no marked change in the latency was manifested in the initial positive response P_1 of the VEP obtained in L_3, but the latencies of P_2 and N_2 were prolonged, and some of the later positive and negative responses were decreased while others were deteriorated (Fig. 14B and C). In the VEP brought out in SS_2, the latency of P_1 also did not show changes, but the later positive and negative deflections were depressed considerably (Fig. 14B). Similar changes were also observed in the VEP of the lateral suprasylvian gyrus SS. Destruction of the optic radiation and cerebral white matter near the GL but without impairment of the GL (Fig. 15A) resulted in only slight diminishment in amplitude in the wave form of the VEP in the lateral gyrus and the lateral suprasylvian gyrus (Fig. 15-B, r-L_3 and r-SS_2). In the control experiment in which the putamen was destroyed, VEPs in the lateral (L_3) and lateral suprasylvian gyrus (SS_3) did not show changes in their wave forms or latencies of the response components by destruction, but rather showed increase in amplitude of some components (Fig. 16A, and B) and the autocorrelograms of background EEGs suggested augmentation of some activities (Fig. 5C).

DISCUSSION

I. *Background EEG activities.*

My results in EEGs of the lateral, posterior sigmoid and lateral suprasylvian gyri are in agreement with those of BRAZIER (1963)[4] in that EEGs led from the lateral, posterior sigmoid and lateral suprasylvian gyri of unanesthetized, immobilized cats did not demonstrate such periodic activities of the alpha rhythm as the damped oscillations of about 10 c/s in autocorrelograms (BARLOW, 1959[1]; BARLOW, BRAZIER and ROSENBLITH, 1952[2], etc.) or the power spectra with peaks at 10 c/s (SATO and KITAJIMA, 1965[17]) that have been noted in man. Hower, a considerable amount of intense rhythmic activities of about

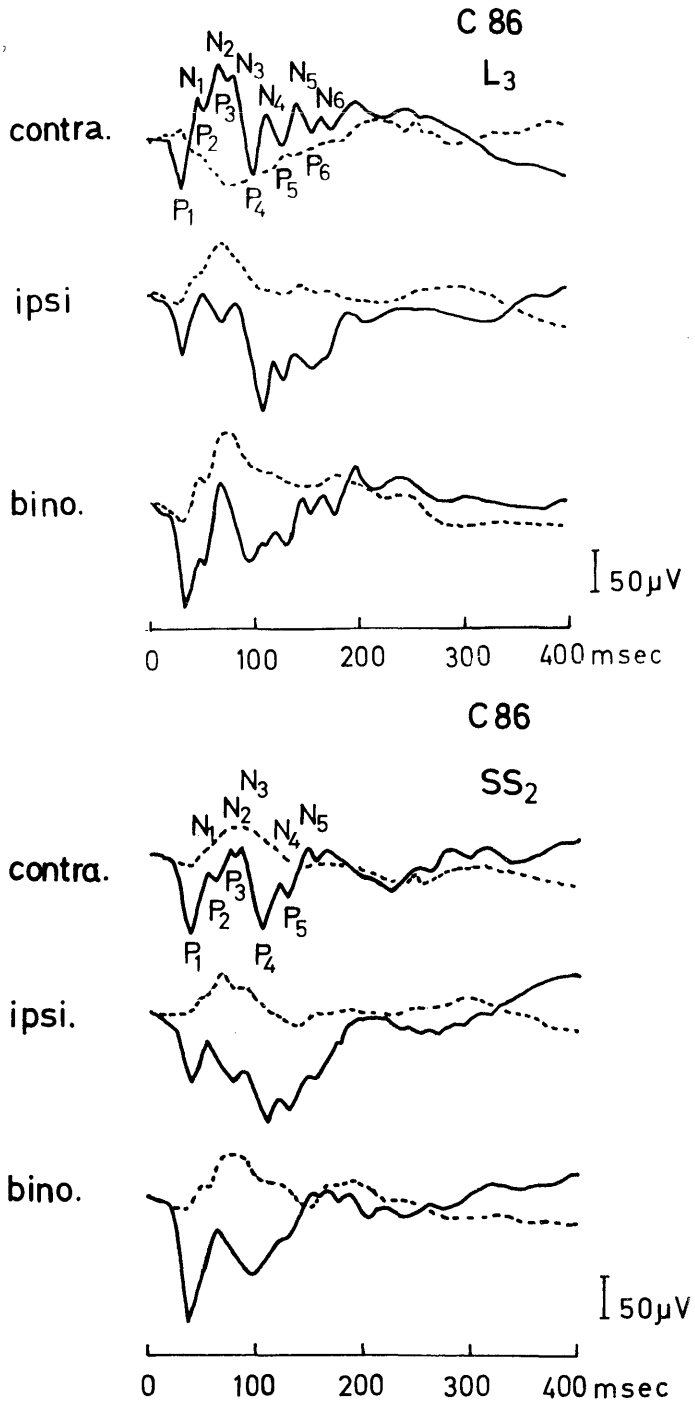


Fig. 12. Simultaneous illustration of VEPs in the visual (L_3) and association areas (SS_2), before and after complete destruction of the lateral geniculate body. Bold and dotted lines: Before and after destruction. Contra., ipsi. and bino.: VEPs due to contra-, ipsi-lateral monocular and binocular flashing light stimulus.

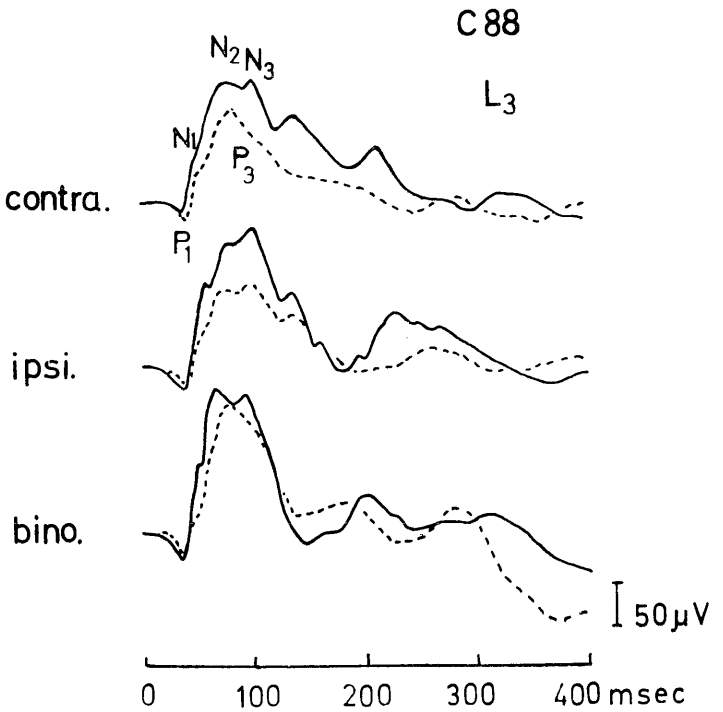


Fig. 13A. VEPs in the visual area (L_3) before and after destruction of GL. Abbreviations, see Fig. 12.

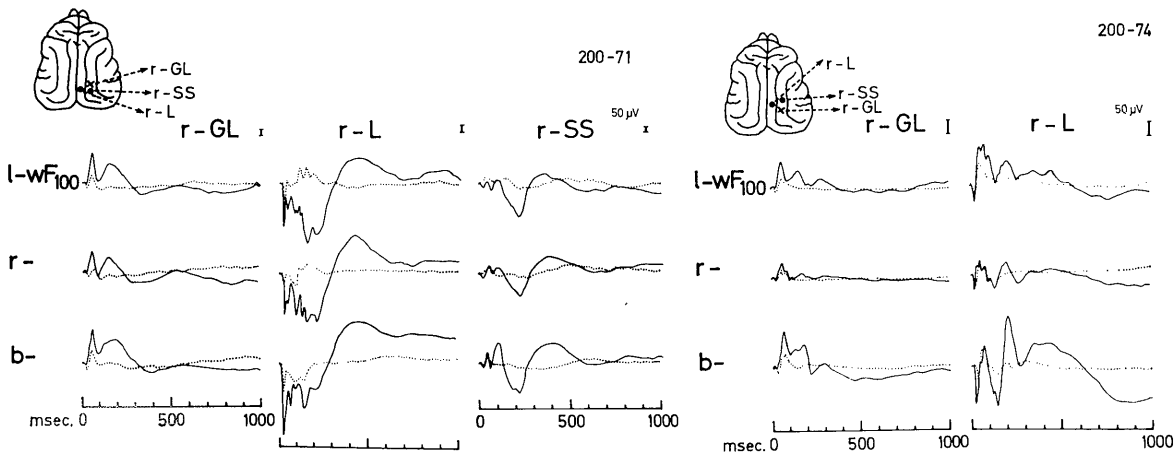


Fig. 13B. Two examples of VEPs before and after destruction of the right lateral geniculate body.

Bold and dotted lines: VEPs before and 20 minutes after destruction. r-GL, r-L, r-SS: right lateral geniculate body, right lateral and right lateral suprasylvian gyri. l-wF_{100}, r-and b-: contralateral, ipsilateral monocular and binocular flashing light stimulus.

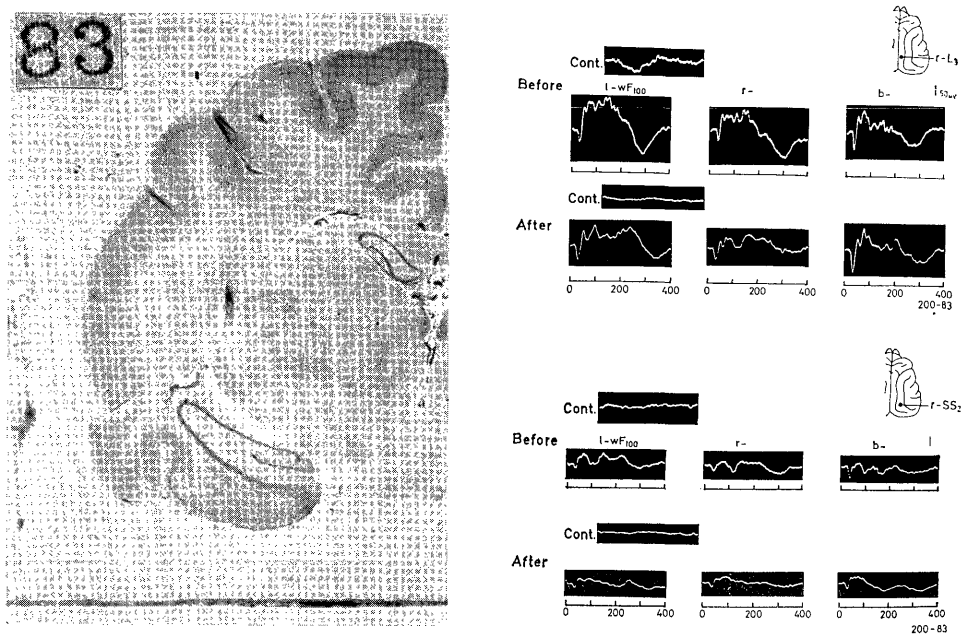


Fig. 14A. (Left) Photograph of transverse section of the brain (cat #83) showing partial destruction of right optic tract and ventrolateral site of the lateral geniculate body.

Fig. 14B. (Right) VEPs of cat #83 before and after destruction in the right visual (Top. r-L_3) and lateral suprasylvian gyri (Bottom, r-SS_2).

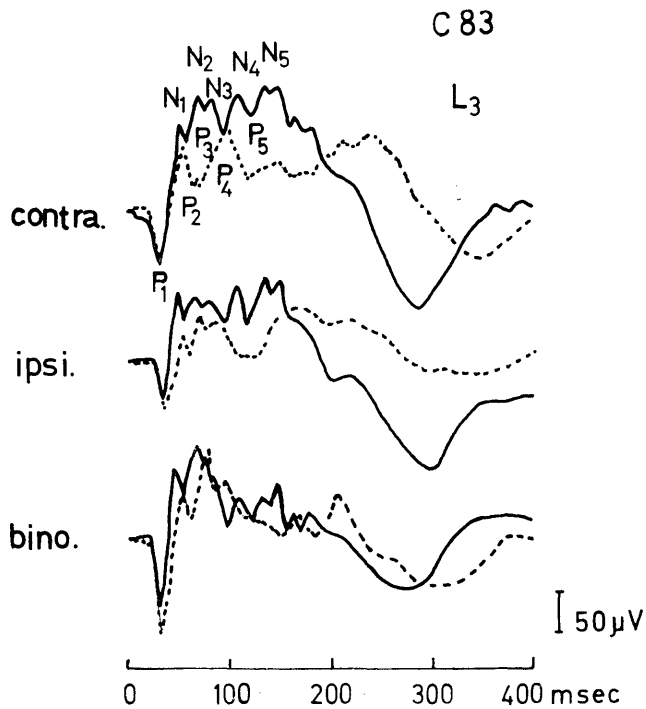


Fig. 14C. Simultaneous illustration of VEPs of cat #83 before and after the destruction.

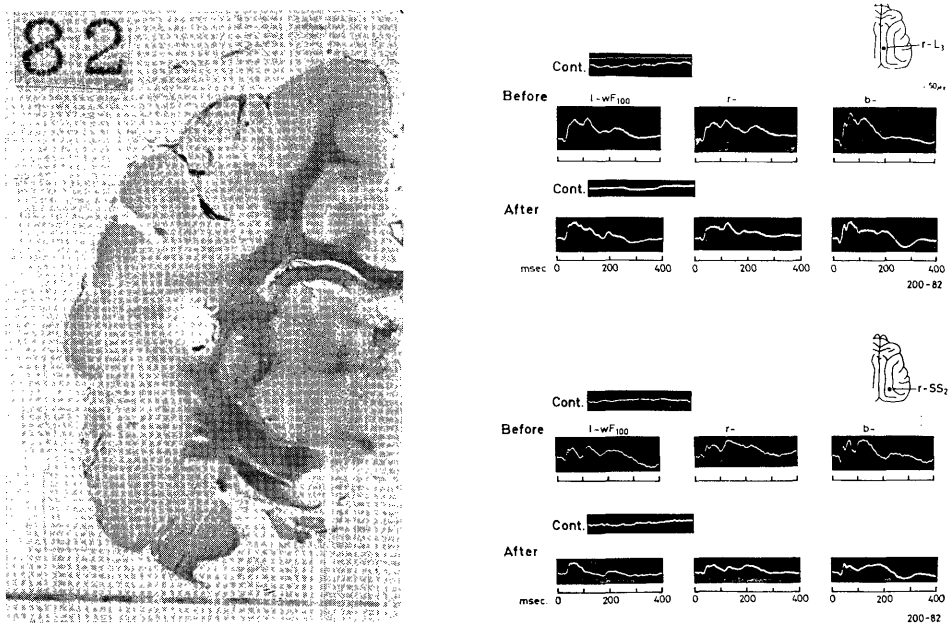


Fig. 15A. (Left) Photograph of transverse section of the brain (cat #82) verifying destruction of right optic radiation and white matter neighbouring the intact lateral geniculate body.

Fig. 15B. (Right) VEPs in the visual (r-L_3) and lateral suprasylvian gyri (r-SS_2) of cat #82.

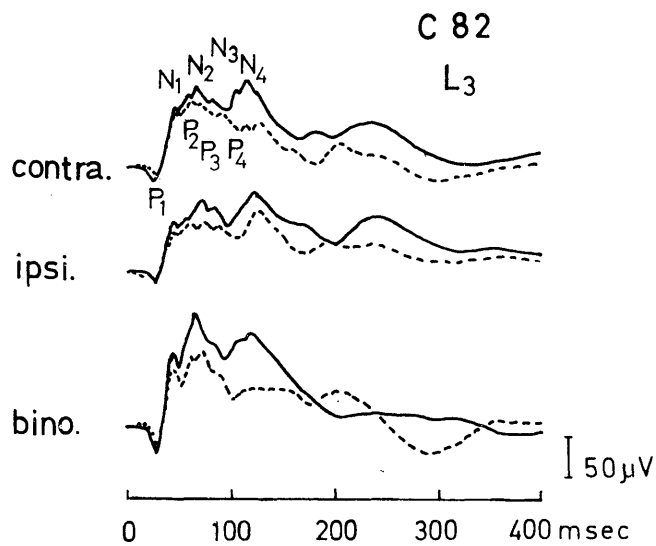


Fig. 15C. Simultaneous illustration of VEPs in L_3 of cat #82 before and after the destruction.

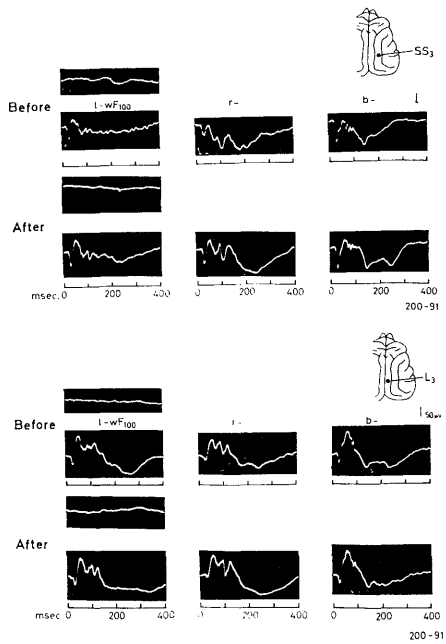


Fig. 16A. VEPs in the lateral suprasylvian (Top, SS_3) and lateral gyri (Bottom, L_3) before and after the destruction of right putamen (cat #91).

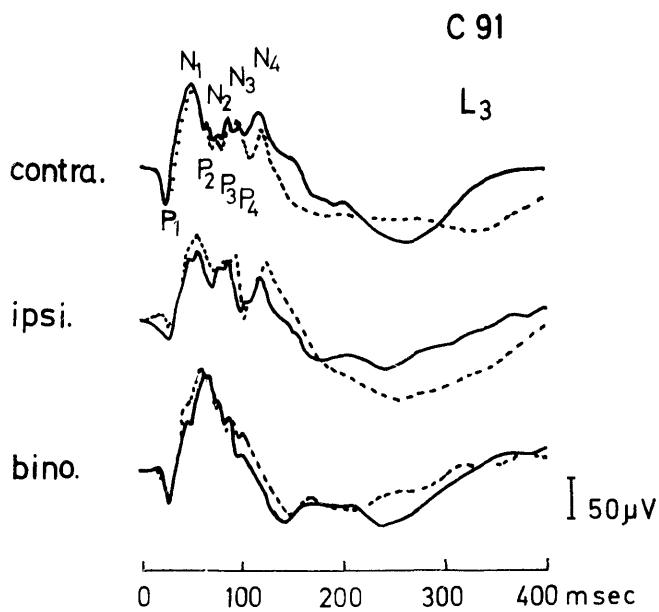


Fig. 16B. Simultaneous illustration of VEPs in L_3 of cat #91 before and after destruction of right putamen.

1−3 c/s were obserevd in most regions of cat and intense activities of about 5 c/s were found to a lesser extent in some regions of some cats, though it was impossible to determine their topographic characteristics. Furthermore, in some cats, oscillations of about 10 c/s activity were superimposed on slower waves in the autocorrelograms of background EEGs before destruction (Fig. 4C), and their power spectra (Fig.4D) showed one or more peaks rising up relatively higher in the frequency range of 8−12 c/s than those in the adjacent frequency ranges. There is a possibility that these activities may have been caused by activities of the visual system, since they were depressed by destruction of a lateral geniculate body on the ipsilateral side. However, evidence was insufficient to permit any speculation on whether they are identical with the alpha rhythm in man.

The fact that depression of EEG activities of less than about 5 c/s frequency in the association area (lateral suprasylvian gyrus) and augmentation of those in the somatosensory area (posterior sigmoid gyrus) were produced by almost complete (Fig. 2C and D) or partial destruction (Fig. 1, C and D) of a lateral geniculate body suggests some intimate interactions between the subcortical specific visual nucleus and these two areas. Presumably, these two interactions act contrary to each other.

II. *Visually evoked potentials (VEP).*

Although STEINBERG (1965)[18] demonstrated eight positive-negative diphasic deflections within 120 msec after the flash stimulus in the VEP led from the lateral gyrus of unrestrained cat, the author was able to observe only four deflections in that of cats immobilized by Flaxedil. This difference may have been caused by Flaxedil application during the experiment and some depressive effect of ether inhalation during the surgical procedures before the experiment. There was, however, no significant difference in the peak latency of the initial positive deflection noted by STEINBERG and that of the author. The fourth negative response N_4 was also the same in peak latency as wave *g*, and the peak latencies of the preceeding negative responses N_1, N_2 and N_3 seemd to correspond to waves *a*, *c* and *f*, respectively, while those of the positive response P_2, P_3 and P_4 correspond to waves 2, 5 and 7 respectively. Following the initial positive P_1 response, a slow negative wave lasting about 120 msec (Fig. 10, C74, 75, 78 and 86, Fig. 11, Type II) was often observed which would be wave *α*.

In the visual cortex of rabbit, MIMURA et al. (1967)[13] have observed a slow negative (*ns*) following the initial positive deflection onto which four positive (-negative) diphasic deflections, $P_2(N_2)$, $P_3(N_3)$, and $P_5(N_5)$, were superimposed. It lasted for about 140 msec with a peak at around 100 msec after delivery of the flash stimulus. Since this

wave appeared when the level of cortical EEG activty is high (Stages I and II) with an EEG pattern of wakefulness, this wave may correspond to wave α in the cat visual cortex. Negative waves similar to α, β and γ, the former two waves having superimposed sharp negative and positive deflections N_1, P_2, N_3, have also been found by OCHI(1968)[15] in the VEP of visual cortex of monkey after removal of the sharp deflections through the method of moving average. The rabbit VEP in the EEG stages of wakefulness seemed to be coincided with that of Type III in cat.

In the rabbit VEP, the slow negative wave *ns* changes to a positive wave with decrease in spindle EEG activity (Stage III) (MIMURA et al. 1967[13]). This may suggest a positive slow wave in the Type IV VEP of cat (Fig. 11), in which spindle weves are observed occasionally in EEG, especially in that of the posterior sigmoid gyrus. Although STEINBERG has observed later slow negative waves, β and γ, and smaller oscilatory potentials, $\triangle 1-\triangle 5$, they were found inconsistently by this author (e.g. Fig.10, C74, etc.), so that superimposition of several VEPs did not reveal regular pattern in the range of the above responses (130−1000 msec in Fig. 10 Bottom), probably because superimposition had been done with VEPs of different types.

NORTON ahd JEWETT[14] have observed eight different wave patterns in the VEPs in the lateral gyrus of unrestrained cats. Their pattern 6 seems to coinside to Type I of the author and pattern 3 to Types II and III, though the latencies of the later responses were longer in the immobilized cats probably due to the acute experiment. However, none of the above eight wave patterns corrsponded to the author's Type IV. In the immobilized cats, VEPs most frequently noted were those of Type II and III, which correspond to wave form 3 which is demonstrated in states of alertness and spindle sleep of unrestrained cats. In the authors acute experiments, background EEGs in the postsigmoid, lateral and lateral suprsylvian gyri showed low voltage fast and/or spindle waves, which may agree with the above result of NORTON and JEWETT.

Although VEPs have previously been demonstrated not only in the cerebral visual area but also in the lateral suprasylvian gyrus (MARSHALL 1943[12], DOTY 1958[8], THOMPSON 1963[20], BIGNALL 1966[5]), deflections in the VEP were marked within 100 msec after the flash stimulus. However, later deflections were noted in the VEP of lateral suprasylvian gyrus (SS_3, SS_2) that were similar to the diphasic ones evoked in the lateral gyrus in their wave form, but tended to be longer in their peak latencies (Fig. 9). In spite of the lack of a definite difference between the initial positive response P_1 led monoporarily in the lateral suprasylvian gyrys (SS_3) and that in the lateral gyrus, the former was found to be longer in duration when the VEPs were led bipolarily (Fig. 7).

This evidence suggests that the train of afferent inflow through the direct pathway from the lateral geniculate body to the lateral suprasylvian gyrus (VASTOLA 1961[22]) caused by a single flash stimulus may require a longer time than that to the lateral gyrus. In addition, the difference in the wave forms of the later deflections (P_2N_2, P_3N_3, etc.) following the initial positive response more prominent than in those obtained monopolarily (Fig.7), and the tendency of longer peak latencies of later responses in the lateral suprasylvian gyrus may suggest that the indirect pathways through the dorsolateral thalamus (BUSER 1959[6]) and/or visual cortex (CLARE 1954[7], IMBERT 1966[10]) and other nonspecific pathways to the lateral suprasylvian gyrus is longer than the indirect pathway to the lateral gyrus.

Complete or almost complete disappearance of the initial positive-negative P_1N_1 and other deflections in the VEPs in the lateral suprasylvian gyrus (Fig.12, SS_2) due to almost complete destruction of the lateral geniculate body may evidence in support of the direct input to this gyrus from this geniculate body as postulated by VASTOLA 1961[22]. The slow wave exhibited after the similar destruction may be the wave α demonstrated by STEINBERG. Since the wave α lasts for about 120 msec after the flash stimulus and its peak is at 70 msec, it may be brought out via indirect input (nonspecific projection system).

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